Acute Heart Valve Emergencies

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KEYWORDS

• Acute aortic regurgitation • Acute mitral regurgitation • CICU • Severe aortic stenosis
• Severe mitral stenosis • Tricuspid regurgitation • Tricuspid stenosis • Valvular emergency

KEY POINTS

- Acute aortic regurgitation results in rapid equilibration of aortic and left ventricular diastolic pressures, which increases left ventricular wall stress and decreases coronary perfusion. Goals of therapy include decreasing the diastolic filling period with reduction in regurgitant volume.
- Severe aortic stenosis resulting in decompensated heart failure or cardiogenic shock is due to a fixed obstruction, which increases left ventricular wall stress and decreases stroke volume. Management includes decreasing the left ventricular to aortic gradient.
- Acute mitral regurgitation results in a precipitous increase in pressure within an unprepared left atrium, and left ventricular ejection fraction may appear seemingly preserved despite cardiogenic shock. Treatment includes optimizing forward flow and decreasing pulmonary edema.
- Severe mitral stenosis results in an elevated left atrial pressure with a normal or low left ventricular end-diastolic pressure. Cardiogenic shock can occur from right ventricular failure with enhanced interventricular interaction.
- For all severe valvular lesions that result in decompensated heart failure or cardiogenic shock, appropriate medical therapy is essential for stabilization, but expeditious correction of the underlying anatomic problem should not be delayed.

Video content accompanies this article at http://www.cardiology.theclinics.com.

INTRODUCTION

Although many patients in the cardiac intensive care unit (CICU) have chronic valvular heart disease, a primary valvular problem accounts for approximately 8% of all admissions.[1] In this setting, valvular emergencies can encompass both acute dysfunction and decompensation due to a progressive valvular lesion. For both scenarios, but especially for acute lesions where compensatory cardiac remodeling has not occurred, prompt recognition, stabilization, and definitive treatment are essential for optimal patient outcomes. Accordingly, the aim of the current article is to highlight commonly encountered acute valvular emergencies with a focus on pertinent clinical and hemodynamic findings, essential echocardiographic features, medical and temporary mechanical circulatory support management, and finally, definitive repair with transcatheter-based intervention or surgery.

AORTIC REGURGITATION

Acute, severe aortic regurgitation (AR) must be promptly recognized to prevent multi-organ failure and hemodynamic collapse. Once cardiogenic shock has occurred, medical options are limited, and emergency intervention is often required.

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Causes

Acute AR can result from infective endocarditis, aortic dissection, prosthetic or allograft valve dysfunction, and trauma/iatrogenic injury.[2] In categorizing the mechanism of AR, the Carpentier classification is often employed: type I (normal leaflet excursion, ie, annular dilation or leaflet perforation), type II (cusp prolapse or flail), and type III (cusp restriction).[3] Infective endocarditis can cause leaflet perforation and/or leaflet flail. In particular with prosthetic valves, perivalvular extension of infection (ie, aortic root abscess) can result in complete or partial valve dehiscence and paravalvular leak. Aortic dissections can result in acute AR through annular dilation leading to incomplete coaptation of the aortic valve leaflets, direct extension of the dissection resulting in a flail leaflet or commissural disruption, and prolapse of the dissection flap into the left ventricular outflow tract (LVOT). Iatrogenic acute AR can result from leaflet perforation or impingement during left heart catheterization, and during balloon valvuloplasty of a stenotic aortic valve. Blunt trauma to the chest is a rare but reported etiology of acute AR.[4] Patients with a bicuspid aortic valve are also at increased risk of developing acute AR due to either infection,dissection, or trauma.

Clinical and Hemodynamic Presentation

In acute AR, there is a rapid influx of blood into the left ventricle (LV) during diastole, leading to an equalization of LV and aortic pressures and a precipitous rise in left ventricular end-diastolic pressure (LVEDP). This initially leads to premature closing of the mitral valve in diastole and incomplete left atrial emptying with resultant pulmonary edema. If the LVEDP continues to increase and exceeds left atrial pressure, diastolic mitral regurgitation (MR) can occur. In addition, effective forward stroke volume is reduced, and in a chronotropically competent patient, heart rate increases in an attempt to maintain cardiac output. An invasive hemodynamic assessment often reveals elevated filling pressures and a low cardiac index. In acute AR, when there has not been time for compensatory LV enlargement, the pulse pressure may be normal or even reduced if the stroke volume is low.

Echocardiographic Diagnosis

AR is initially assessed with transthoracic echocardiography (TTE), although transesophageal echocardiography (TEE) is often required to better delineate the anatomic mechanism of AR and to aid in surgical/procedural planning.[3,5–7] On TTE, color flow Doppler can be used to identify the presence or absence of AR, particularly in a parasternal long or apical 5-chamber view. The ratio of the AR jet width to LVOT diameter is a simple, relatively sensitive screen for AR. However, this methodology can underestimate eccentric jets of AR and is affected by the diameter of the LVOT. A vena contracta can also be measured while in a parasternal long axis view and can help delineate mild (VC < 0.3 cm) from severe AR (>0.7 cm). This method is also prone to measurement error and is less reliable with bicuspid aortic valves.

Continuous wave Doppler can be used to evaluate the severity of acute AR. The pressure halftime (PHT) is a measure of equalization of pressures between the LV and aorta, and a PHT less than 200 m sec is compatible with severe AR.[3,5] However, this parameter reflects the time course of AR development more than the severity. Specifically, in acute severe AR, when the LV has not had time to remodel to accommodate the increased LVEDP, the PHT will be short. Conversely, in chronic severe AR with LV remodeling, the PHT is not a reliable measure of AR severity.

The presence of flow reversal in the thoracic and abdominal aorta is another useful semiquantitative parameter to assess the severity of AR. Any degree of diastolic flow reversal in the abdominal aorta is compatible with severe AR. Of note, in acute severe AR, due to the rapid equalization of pressures between the LV and aorta, flow reversal may not be holo-diastolic. Finally, assessment of the LV response is crucial. In acute AR, left ventricular size is normal, and LV ejection fraction may be reduced because of increased wall stress and decreased coronary perfusion pressure.

Medical Management and Mechanical Support Options

In acute severe AR, urgent medical stabilization is critical to prevent further deterioration. A cornerstone of medical management in AR is reducing diastolic filling time to maintain cardiac output and reduce LVEDP. The avoidance of bradycardia is essential, and even a normal sinus rhythm (ie, 70 beats/min) may be inadequate. Temporary transvenous pacing or inotropes should be considered in patients with acute AR with bradycardia or normal sinus rhythm who have acute decompensated heart failure or cardiogenic shock. In patients with AR in the setting of an aortic root abscess who are at high risk of progression to complete heart block, which can precipitate rapid hemodynamic collapse, temporary pacing can also be considered. Atrioventricular nodal blockade should be avoided in patients with acute

severe AR, including patients with an acute ascending aortic dissection.[7]

Afterload reduction with intravenous (IV) vasodilators can reduce the regurgitant flow and help stabilize patients.[7] Preload reduction with diuretics can also aid in reducing pulmonary edema. Mechanical circulatory support (MCS) in acute or severe AR is relatively contraindicated as currently available MCS options require a competent aortic valve. An intra-aortic balloon pump will worsen AR regurgitant volume. An Impella device (AbioMed, Danvers, MA), which must cross the aortic valve and rests in the LV, will also worsen AR due impaired leaflet coaptation (and possible leaflet damage). Veno-arterial extracorporeal membrane oxygenation (ECMO) increases LV afterload, LV wall stress, and results in pulmonary edema. An additional venous cannula can be placed in the left atrium to attenuate pulmonary edema (LAVA ECMO). However, this approach is reserved for the rare patients in extremis who are not currently candidates for emergency intervention, but definitive repair is planned in the near-term.

Definitive Repair

Surgical repair or aortic valve replacement (SAVR) carries a class I indication in acute, severe AR (**Fig. 1**, Videos 1–3).[7] In patients with decompensated heart failure or cardiogenic shock, surgical replacement should not be delayed unless the intraoperative risk is prohibitive. Transcatheter aortic valve replacement (TAVR) for severe AR has traditionally been considered a contraindication in native valves, as earlier generation TAVR valves were associated with valve embolization and paravalvular leak (due to a dilated aortic annulus). However, newer generation TAVR valves have shown an improvement in clinical outcomes (**Fig. 2**, Videos 4–6).[7–9] Moreover, transcatheter

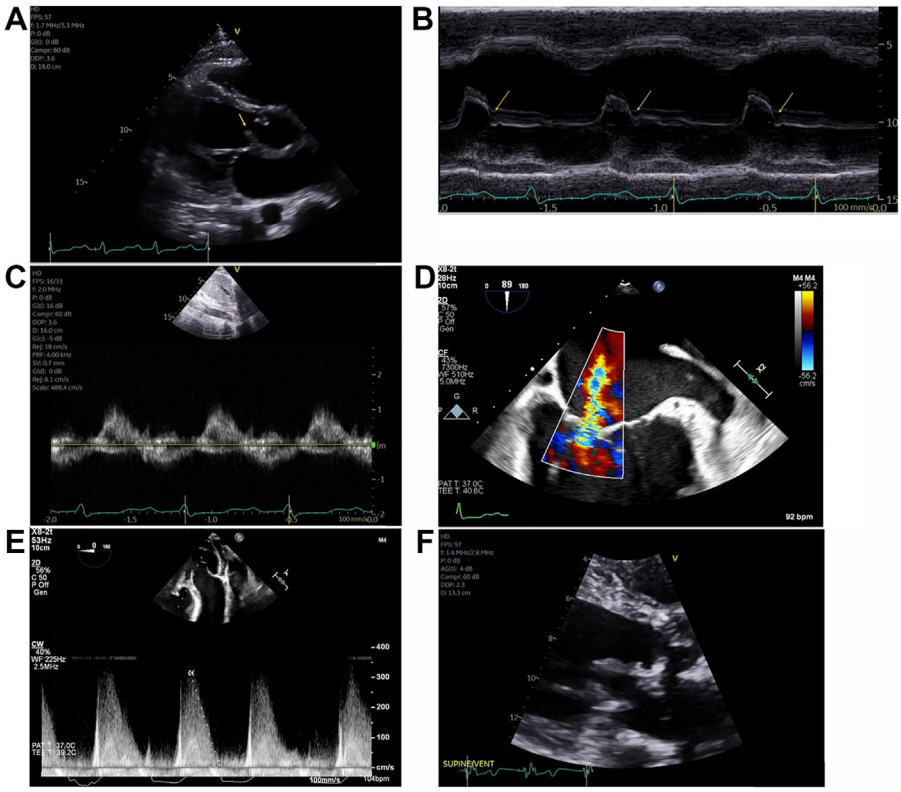

Fig. 1. 40-year-old intravenous drug user with group B streptococcus bacteremia presenting with hypotension. (*A*) Parasternal long-axis view shows a flail non-coronary cusp, and pre-mature closure of the mitral valve in keeping with elevated left ventricular diastolic pressure. (*B*) M-mode through the mitral valve from a parasternal long-axis view confirms premature closure (*arrows*). (*C*) Diastolic flow reversal in the abdominal aorta compatible with severe aortic regurgitation. (*D*) Transesophageal echo shows diastolic mitral regurgitation in the setting of acute severe aortic regurgitation, reflective of increased left ventricular diastolic pressure. (*E*) Short pressure half-time consistent with rapid equilibration of aortic and left ventricular diastolic pressures. (*F*) Patient underwent successful aortic valve replacement with a bioprosthesis as well as mitral valve repair including repair of aortic-mitral continuity.

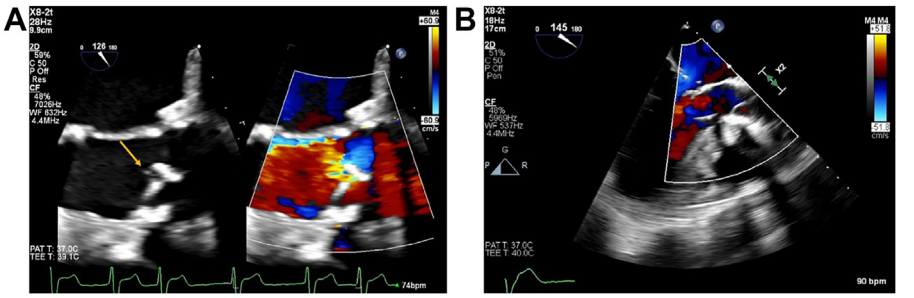

Fig. 2. 60-year-old with multiple prior cardiac surgeries, most recently with aortic valve replacement with a homograft as well as mitral valve repair with an annuloplasty band and reconstruction of the aortic-mitral continuity presents with (*A*) flail of the anterior cusp (*arrow*) and eccentric posteriorly directed aortic regurgitation, noted on long-axis view of the aortic valve with TEE. (*B*) Status post 29 mm Edwards S3 valve within the aortic homograft with no residual aortic regurgitation.

aortic valves designed specifically for AR are currently being investigated in clinical trials.

AORTIC STENOSIS

In the elderly population, aortic stenosis (AS) is the most prevalent valvular heart disease.[10,11] The most common causes are calcific and congenital AS (ie, due to a bicuspid aortic valve). When severe AS results in decompensated heart failure or cardiogenic shock, urgent or emergency intervention is required.

Clinical and Hemodynamic Presentation

On exam, patients with severe AS have a late-peaking crescendo-decrescendo systolic murmur with a diminished or absent A2 and *pulsus parvus et tardus*. In the setting of decompensated heart failure, patients with severe AS will have elevated biventricular filling pressures and may have a reduced cardiac index. In a tenuous patient, invasive hemodynamic monitoring is essential because the fixed obstruction from AS renders the patient sensitive to shifts in preload and afterload.

Echocardiography

The initial assessment of AS severity is based on peak jet velocity, mean transvalvular gradient, aortic valve area, and the dimensionless index (DI).[12] Peak jet velocity and mean gradient are measured using continuous wave Doppler in

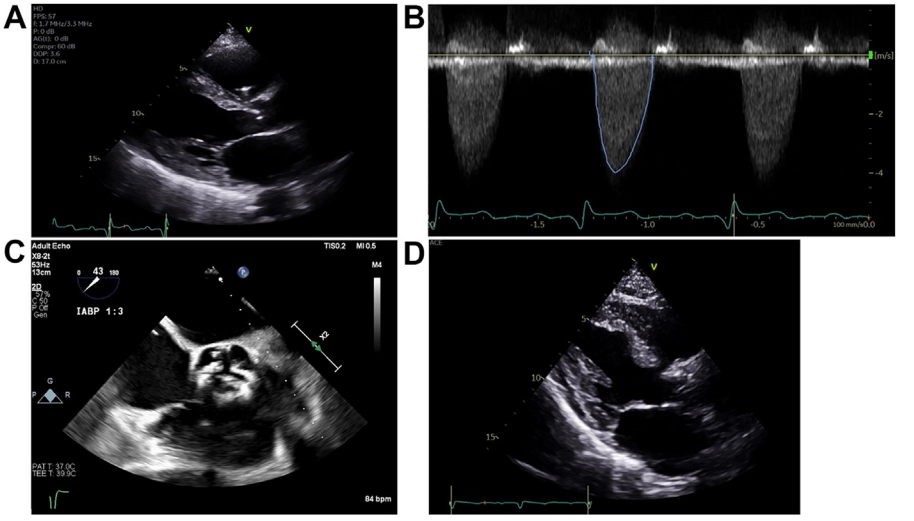

Fig. 3. 70-year-old presents with cardiogenic shock. (*A*) Parasternal long axis view shows severe left ventricular systolic dysfunction with thickened aortic valve leaflets. (*B*) Continuous wave Doppler shows a peak gradient of 63 mm Hg and a mean gradient of 37 mm Hg compatible with severe aortic stenosis. (*C*) Short axis transesophageal echo shows a trileaflet aortic valve with limited leaflet excursion. The patient was supported with an intra-aortic balloon pump (IABP) for cardiogenic shock. (*D*) The patient underwent transcatheter aortic valve replacement with a 26 mm Edwards S3 valve with improvement in left ventricular systolic function.

multiple acoustic windows to obtain the highest velocity. A peak velocity of greater than 4.0 metre per second and a mean gradient of greater than 40 millimetre Hg is consistent with severe AS, whereas a velocity of greater than 5.0 metre per second indicates very severe AS. AS severity is also assessed with aortic valve area (AVA), and an AVA of less than 1.0 square centimetre (<0.6 cm^2/m^2) is compatible with severe AS. Finally, the ratio of the LVOT and AV velocity time integrals (DI or velocity ratio) is standard in the assessment of AS, and a ratio of less than 0.25 is in keeping with severe AS.

In patients with a reduced stroke volume, transvalvular gradients may not reach classic severe thresholds despite severe AS (low-flow, low-gradient (LFLG)). In this setting, the AVA and DI may be more informative, and further imaging with TEE or computed tomography can aid in confirming severe AS. Moreover, as AS progresses, the time to peak velocity (acceleration time [AT]) increases, analogous to the physical exam finding of *pulsus tardus*. Therefore, an increased AT, or an elevated AT to ejection time ratio, is associated with AS severity. Similarly, in patients with prosthetic valves, severe stenosis is suspected when

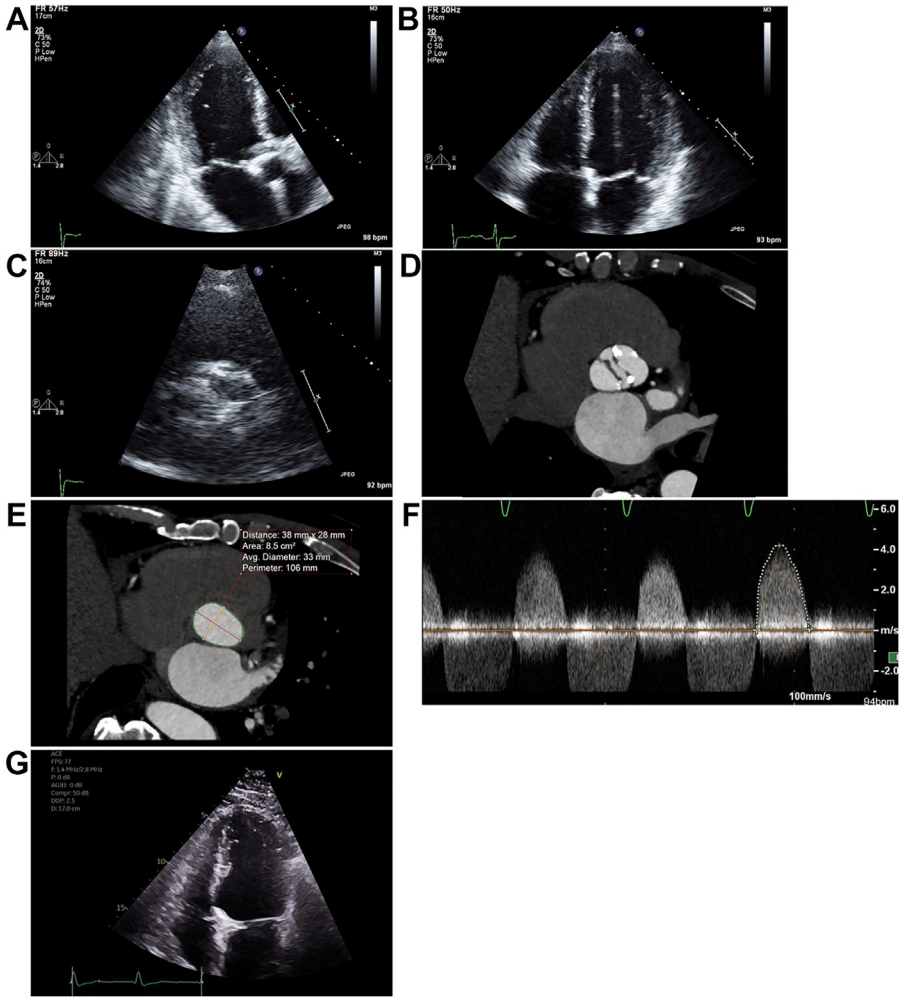

Fig. 4. 65-year-old presents with decompensated heart failure in setting of aortic stenosis. (*A*) Apical long axis view shows severely reduced left ventricular systolic function with hyperechoic and thickened aortic valve leaflets with decreased excursion. **(***B*) Apical 4 chamber view show reduced left and right ventricular systolic function. (*C*) Aortic valve morphology is difficult to delineate on this short-axis view. (*D*) 4D CTA showed a bicuspid aortic valve with asymmetric calcification of the sinuses. (*E*) On CTA, the aortic annulus was enlarged, which favored surgical versus transcatheter aortic valve replacement. (*F*) Continuous wave Doppler from the right upper sternal border is compatible with severe aortic stenosis with a prolonged acceleration time and increased acceleration time (AT) to ejection time (ET) ratio. (*G*) Left and right ventricular systolic function is improved after surgical aortic valve replacement.

the DI is less than 0.25 and the AT is greater than 100 metre per sec.[13]

Medical Stabilization and Mechanical Support Options

Medical stabilization focuses on optimizing afterload and preload. Nitroprusside can be useful in reducing the LV to aorta gradient and improving cardiac output,[14] though invasive hemodynamic monitoring is essential because overly zealous treatment can result in hypotension and reduced coronary perfusion. With preload, an adequate LV end-diastolic volume is necessary to maintain an acceptable stroke volume. However, compensatory remodeling from AS reduces LV compliance and excess preload will result in pulmonary edema and pulmonary hypertension. Reducing preload with IV diuresis can ameliorate pulmonary edema, but over-diuresis can lead to a reduction in stroke volume and hypotension.

Regarding MCS options in a patient with cardiogenic shock despite medical therapy, an IABP will reduce afterload and improve coronary perfusion. Few data are available regarding percutaneous left ventricular assist devices, such as the Impella, and there is a manufacturer recommendation against its use with an AVA of less than 0.6 square centimetre. Finally, venoarterial extracorporeal membrane oxygenation (VA-ECMO) may be considered to support end-organ perfusion in refractory shock. However, the fixed obstruction increases LV afterload as well as the risk for aortic valve and LV thrombus, and expeditiously correcting the valvular stenosis is indicated.[15]

Definitive Repair

With decompensated heart failure or cardiogenic shock due to AS, aortic valve replacement (AVR) carries a class 1A indication.[7] Given the limitations of medical therapy, unless AVR is prohibitive-risk, definitive treatment should not be delayed. If the anatomy is amenable to TAVR, this approach has emerged as a good treatment option for the extreme-risk patient (**Fig. 3**, Videos 7–9).[16] Importantly, for patients with embarrassed left ventricular systolic function due to high gradient AS, rapid improvement in left ventricular ejection fraction (LVEF) is expected after AVR with relief of the afterload mismatch. In a situation, where there is uncertainty regarding the contribution of AS to the clinical deterioration, percutaneous balloon aortic valvuloplasty (PBAV) can be a diagnostic as well as a therapeutic measure.[7,17–19] However, PBAV is contraindicated in patients with moderate or greater AR.

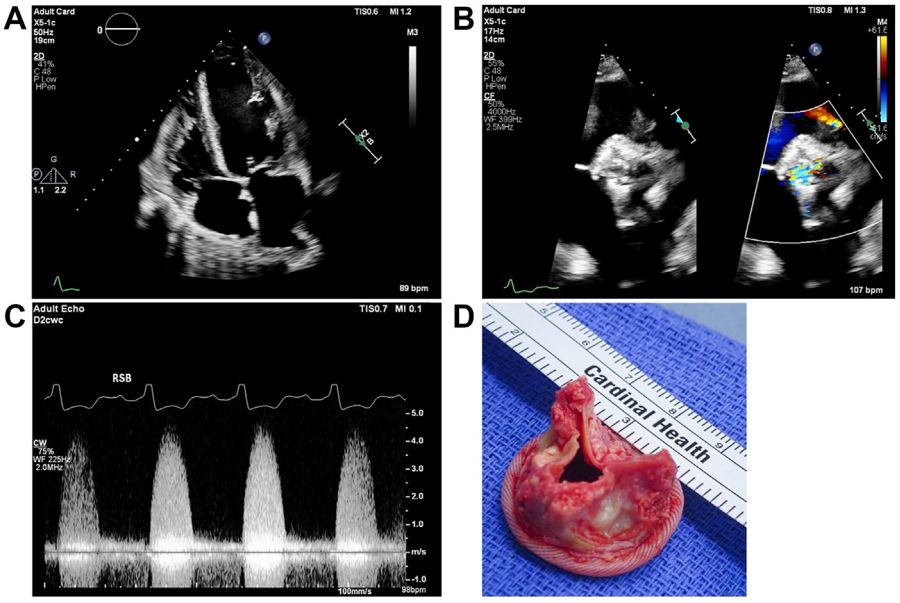

Fig. 5. 38-year-old with a history of congenital aortic stenosis and two prior cardiac surgeries, most recently with bioprosthetic aortic valve replacement, presented in cardiogenic shock. (*A*) Apical 4 chamber view shows severe left and right ventricular systolic dysfunction. Given progressive cardiogenic shock, she was placed on VA ECMO as a bridge to cardiac surgery. (*B*) Short axis view of the bioprosthetic aortic valve with color compare shows hyperechoic immobile leaflets. (*C*) Continuous wave Doppler from the right upper sternal border (RSB) shows peak and mean gradients of 76 and 44 mm Hg, respectively. (*D*) Given the small annulus and valve, she underwent surgery. The bioprosthetic leaflets were severely calcified and immobile with no signs of infection.

Patients with bicuspid aortic valves present a unique challenge.[20] Specifically, anatomic considerations such as an enlarged annulus, asymmetric sinuses, or raphae calcifications may make TAVR less favorable (**Fig. 4**, Videos 10–14). In addition, these patients may have an associated aortopathy that warrants surgical correction. Therefore, many of these patients should undergo surgery unless the risk is prohibitive. Anatomic considerations are also paramount for decision-making in patients with prosthetic AS. For example, patients with decompensated heart failure or cardiogenic shock may preferentially undergo TAVR unless there are concerns such as a small annulus, potential coronary artery obstruction, or active infective endocarditis (**Figs. 5** and **6**, Videos 15–17).

ACUTE MITRAL REGURGITATION

Similar to acute AR, when an acute onset has precluded gradual compensatory remodeling of the left-sided chambers, severe MR can result in florid pulmonary edema and cardiogenic shock, which requires emergency intervention.

Causes

In general, MR is classified according to whether the valvular pathology is primary or secondary, and the Carpentier methodology is also employed: type I (normal leaflet excursion), type II (leaflet prolapse or flail), and type III (restricted leaflet motion). With myocardial ischemia, acute MR can occur through 2 mechanisms. First one is posterior papillary muscle rupture (either partial or complete) and is generally seen with an inferior myocardial infarction because the posterior papillary muscle has a single arterial supple, unlike the anterior papillary muscle.[21,22] The second mechanism is restricted posterior leaflet motion due to regional wall motion abnormalities, again most commonly encountered with an inferior myocardial infarction. Non-ischemic acute MR can result from chordal rupture with leaflet prolapse and flail in the setting of degenerative mitral valve disease and may also occur due to infective endocarditis.

Clinical and Hemodynamic Presentation

In acute severe MR, the left atrial pressure increases precipitously, and forward stroke volume dramatically decreases. The result is a decreased transmitral gradient and potential underestimated of MR on both echo and auscultation (ie, “silent MR”). With invasive hemodynamic monitoring, although no feature is pathognomonic for acute severe MR, pulmonary hypertension, and an increased pulmonary capillary wedge pressure with tall v waves are observed.

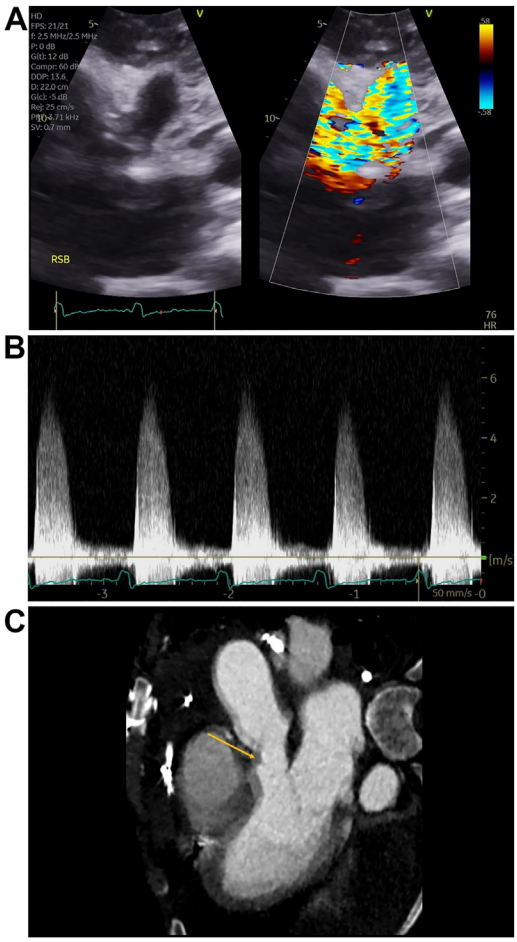

Fig. 6. 55-year-old presented in cardiogenic shock after recent aortic homograft placement at an outside institution for infective endocarditis. (*A*) Echocardiography at the right upper sternal border shows turbulent flow across the homograft. (*B*) Continuous wave Doppler at the right upper sternal border shows peak and mean gradients of 138 and 76 mm Hg, respectively. (*C*) On CTA, there was suggestion of kinking of the proximal aspect of the aortic homograft, which was confirmed at the time of repeat cardiac surgery.

Echocardiography

When acute MR is suspected, echocardiography should be performed at the bedside with the goals of classifying severity, assessing LV response, and delineating MR mechanism.[23] Oftentimes, TEE is necessary to expeditiously achieve these objectives.[3] Color flow imaging can be useful for an initial assessment of the size, geometry, and direction of the MR jet. In acute severe MR, continuous Doppler demonstrates a dense triangular profile

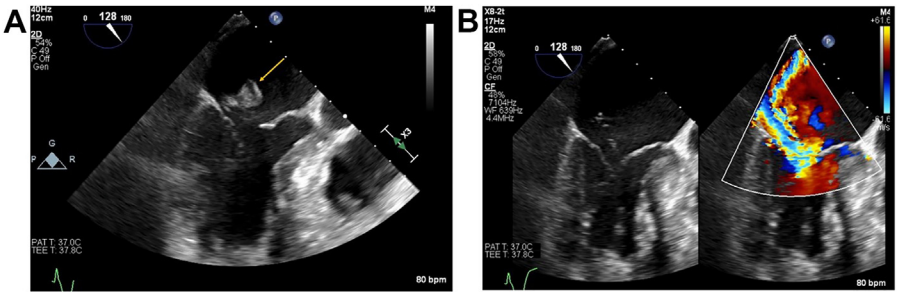

Fig. 7. 78-year-old presented with inferior STEMI, distal RCA occlusion, and cardiogenic shock. Given hemodynamic instability, emergency transesophageal echocardiogram was performed (*A*) which showed rupture of the posteromedial papillary muscle (*arrow*). (*B*) Left ventricular ejection fraction was preserved, which is often the case with acute papillary muscle rupture, and there was severe mitral regurgitation.

indicating rapid equilibration of left ventricular and atrial pressures. Importantly, if the left atrial pressure is markedly elevated and/or systolic blood pressure is low, the MR jet will have a low velocity reflective of a decreased gradient between left ventricular and atrial pressures. On mitral inflow, the E velocity is generally elevated (>1.2 m/sec) indicating increased flow.[3]

Additional measures of MR focus on calculating the regurgitant volume and effective orifice area, although these methods may be technically challenging. The proximal isovelocity surface area (PISA) method is most commonly employed, and in general, an effective regurgitant orifice area (EROA) of greater than 0.4 square centimetre is consistent with severe MR. However, this method is not reliable with multiple or eccentric jets. Moreover, the simplified PISA equation ($r^2/2$) assumes an LV to left atrial pressure gradient of 100 millimetre Hg and should be avoided in the acute setting, where this may not be accurate.

Medical Stabilization and Mechanical Circulatory Support Options

Prompt medical stabilization focuses on reducing pulmonary edema and increasing forward stroke volume. Intravenous vasodilator therapy, such as with nitroprusside, which is rapidly titratable and has a short half-life, and fluid removal are mainstays of acute management. Unfortunately, acute MR can present with hypotension and MCS may be needed for stabilization. Intra-aortic balloon counter pulsation can be utilized to reduce afterload and improve coronary perfusion.[15] Similarly, a percutaneous temporary LVAD such as the Impella (Abiomed, Danvers, and MA) can be used to offload the LV and reduce left atrial pressure.[24] Venoarterial extracorporeal membrane oxygenation may be indicated with profound cardiogenic shock or when biventricular support is necessary. In this setting, given the increase in LV wall stress and potential worsening of pulmonary edema, an LV vent (such as with an Impella or IABP) is often employed.[25]

Definitive Repair

Acute MR with decompensated heart failure and/or cardiogenic shock requires immediate surgical intervention (**Figs. 7–9**, Videos 18–25). With papillary muscle rupture, mitral valve replacement is more common that mitral valve repair (see **Fig. 7**).[26] For ischemic MR, although replacement may be generally preferred, the decision is more nuanced and should account for the patient's age and expected prognosis, as well as the potential

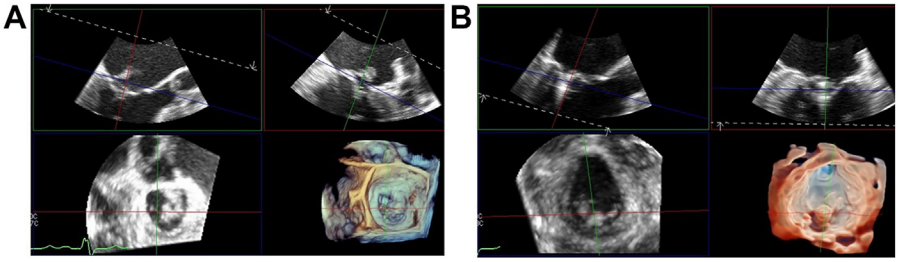

Fig. 8. 50-year-old with a history of coronary artery disease presented with decompensated heart failure and cardiogenic shock. (*A*) 3D multiplanar reconstruction shows rupture of the posteromedial papillary muscle. **(***B*) Due to medical comorbidities including cirrhosis, the patient underwent successful transcatheter edge-to-edge mitral valve repair.

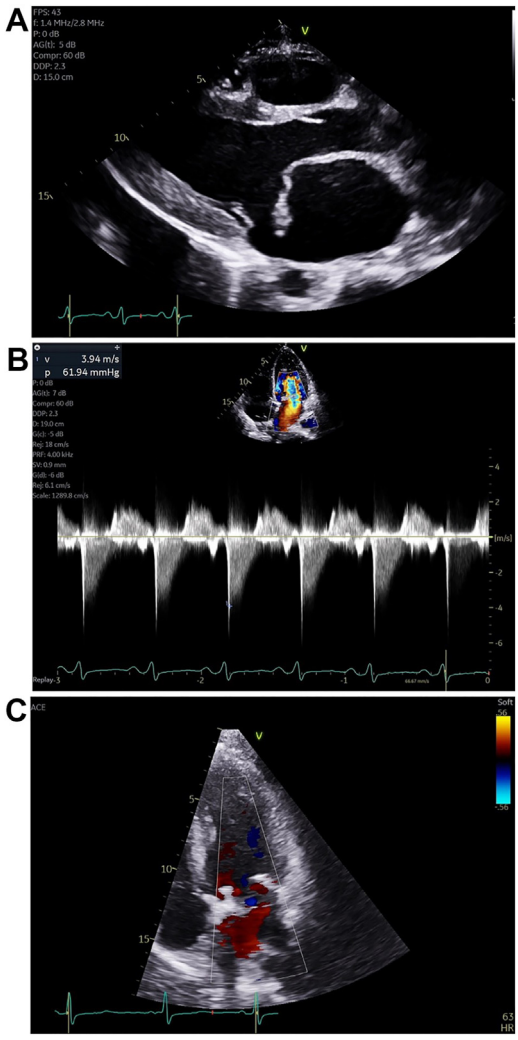

Fig. 9. 39-year-old with oral streptococcal bacteremia presented with cardiogenic shock, acute kidney injury, ischemic hepatitis, and disseminated intravascular coagulation. (*A*) Echocardiogram shows an echodensity associate with the mitral valve, compatible with a vegetation, and a large coaptation defect consistent with severe mitral regurgitation. (*B*) On continuous wave Doppler assessment of the mitral regurgitation, the jet has an early peak and is low velocity. These findings are reflective of a rapid equalization of systolic pressures between the left ventricle and left atrium, as well as markedly increased left atrial pressure with systemic hypotension. (*C*) Given the degree of multiorgan failure, he was initially supported with VA ECMO, and then underwent bioprosthetic mitral valve replacement with good result.

for LV remodeling with surgery. For both ischemia MR and papillary muscle rupture, transcatheter edge-to-edge repair (TEER) has been reported, and should be considered when operative risk is extremely high or prohibitive (see Fig. 8).[27]

SEVERE MITRAL STENOSIS

In the developing world, mitral stenosis (MS) is most commonly due to rheumatic heart disease.[28] The most common cause of non-rheumatic MS is mitral annular calcification (MAC) which encroaches on the leaflets and restricts motion.[29–31] In patients with prosthetic mitral valves, stenosis can result from structural valve degeneration, thrombosis, or endocarditis. With the exception of prosthetic valve thrombosis and endocarditis, MS does not generally occur acutely, and presentation to theCICU with MS as the primary problem may reflect a lack of appropriate follow-up or a missed diagnosis.

Clinical and Hemodynamic Presentation

Patients with severe MS will have elevated left atrial pressures with a low or normal LVEDP. With this fixed obstruction, a prolonged diastolic filling period will decrease the gradient between the left atrium and ventricle and reduce pulmonary edema. However, when a patient develops cardiogenic shock due to severe MS, this presentation is often the result of a right ventricle that is failing because of pulmonary hypertension.

Echocardiographic Assessment

Measurement of the mitral valve area (MVA) is an essential step in the assessment of MS. MVA can be measured directly with planimetry, though accurate assessment at the leaflet tips is necessary.[32] The MVA can also be calculated via the PHT, and even though there are limitations of this approach, a prolonged PHT is associated with more significant MS. The mean gradient is also correlated with MS severity, and a mean gradient of greater than 10 millimetre Hg is compatible with severe MS. However, the mean gradient is dependent on flow (ie, stroke volume) and heart rate, and both of these parameters may be perturbed for a patient in the CICU.

The downstream consequences of MS should also be well interrogated. The left atrium is invariably enlarged, and estimated pulmonary pressures are elevated. Right ventricular systolic function may also be impaired. In the setting of right ventricular pressure overload, the interventricular septum may shift to the left during systole, and this interventricular interaction can also negatively impact stroke volume.

Medical Stabilization and Mechanical Support Options

MS is a fixed obstruction that results in reduced LV diastolic filling. Medical stabilization balances optimizing left ventricular diastolic volume and

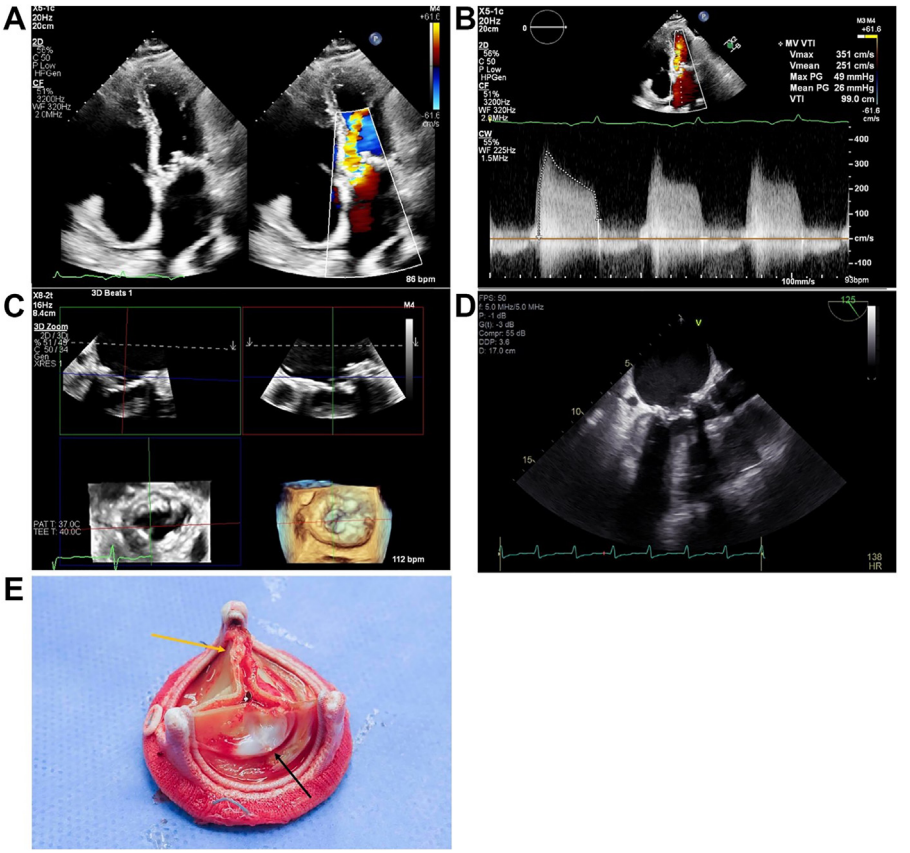

Fig. 10. 50-year-old with a history of an inferior myocardial infarction complicated by papillary muscle rupture status post bioprosthetic mitral valve replacement 10 years prior is admitted with cardiogenic shock. (*A*) Apical 4 chamber view shows thickened, hyperechoic mitral valve leaflets with aliasing on color flow map across the mitral inflow, as well as a dilated and dysfunction right ventricle. (*B*) Continuous wave Doppler of the mitral inflow shows markedly elevated peak and mean gradients at 49 and 26 mm Hg, respectively, with a prolonged pressure half-time (~320 msec) compatible with severe mitral stenosis. (*C*) 3D multiplanar reconstruction shows severely thickened mitral valve leaflet with limited opening during diastole. (*D*) Marked shift of the interventricular septum was noted which contributed to an underfilled left ventricle and raised concern about left ventricular outflow tract obstruction if valve in valve transcatheter therapy was pursued. (*E*) Patient underwent repeat mitral valve replacement. The explanted bioprosthesis showed severe calcification (*orange arrow*) and pannus (*black arrow*) restricting leaflet motion.

reducing pulmonary edema. Decreasing heart rate is often essential in achieving this balance by increasing diastolic filling time. For this reason, beta-blockers are often used, though may be contraindicated in a patient with cardiogenic shock with a prominent component of right ventricular failure. When atrial fibrillation develops, hemodynamic decompensation may occur due to the loss of atrial contraction, and restoration of sinus rhythm should be pursued if feasible. In severe MS, the LV is typically underfilled and vasodilators should be avoided as they may further decrease preload and result in hypotension.

Mechanical circulatory support options are limited in MS. An IABP would provide minimal benefit in augmenting cardiac output, and successful placement of a percutaneous LVAD such as an Impella wound not benefit an underfilled LV. Venoarterial extracorporeal membrane oxygenation with an left artrial (LA) vent (LAVA-ECMO) or a TandemHeart would be feasible MCS options in offloading LA pressure and providing adequate end-organ perfusion, but should only be considered when needed as a short-term bridge to definitive repair.

Definitive Repair

For rheumatic MS, percutaneous mitral balloon valvuloplasty or surgery are considered standard of care.[33,34] With stenosis due to MAC, surgery is first-line, though transcatheter based procedures are promising. If the anatomy is favorable, transcatheter valve-in-valve therapy is a good treatment option for the critically ill patient with

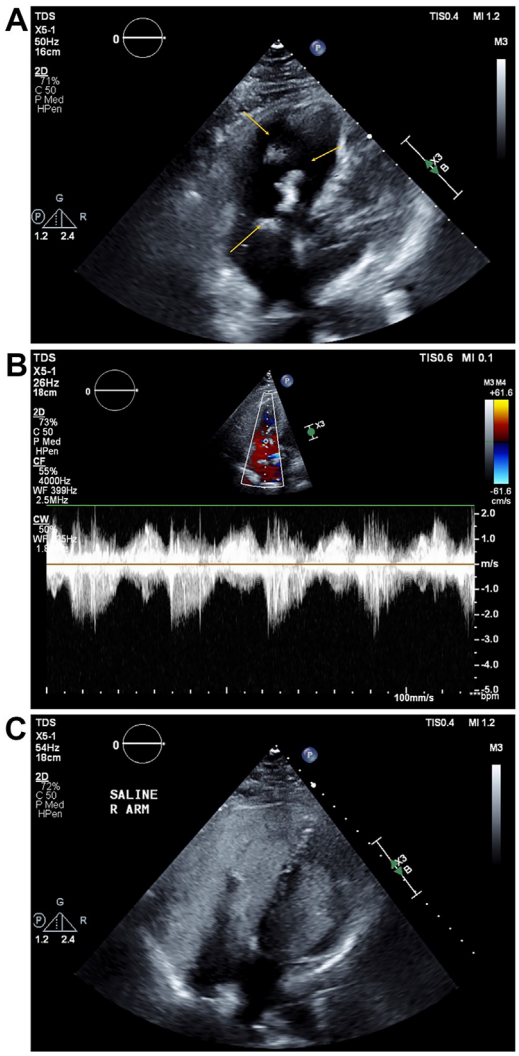

Fig. 11. 38-year-old admitted with refractory hypoxemia. (*A*) Apical 4 chamber view with a focus on the right ventricle shows multiple hypodensities (*arrows*) associated with an intracardiac device lead, which was known to be fractured. The right ventricle is dilated and dysfunctional. (*B*) On continuous wave Doppler assessment, there is a dense and triangular profile compatible with severe, laminar TR. (*C*) On agitated saline study, there is a large right to left shunt across a patent foramen ovale in the setting of elevated right sided pressures and severe TR.

MS due to small vessel disease (SVD. Otherwise, surgery is preferred (**Fig. 10**, Videos 26–28) and is also indicated in patient with prosthetic valve thrombosis or endocarditis.

SEVERE TRICUSPID AND PULMONIC LESIONS

Causes

Severe tricuspid regurgitation (TR) is often a result of other cardiac conditions and can occur from secondary to annular dilation. Primary TR can result from trauma (such as impingement by pacemaker lead placement, or flail leaflet from an endomyocardial biopsy), myxomatous degeneration or from carcinoid syndrome causing leaflet fixation, and lack of coaptation. Native tricuspid stenosis (TS) is rare and is usually caused by mechanical obstruction from an intracardiac mass, such as a tumor or a vegetation. Tricuspid stenosis can also occur with prosthetic valves due to endocarditis, thrombus, or SVD. Pulmonic stenosis (PS) is most commonly congenitally acquired as an isolated lesion or as part of a syndrome such as Tetralogy of Fallot.

Clinical Presentation and Hemodynamics

Severe tricuspid or pulmonic valve disease can present with signs and symptoms of right heart failure including lower extremity edema, ascites, congestive hepatopathy, and cardiorenal syndrome. In severe TR, measurement of right atrial (RA) pressure will demonstrate elevated mean pressures, a steep *y* descent, and potential ventricularization of RA pressures.[35,36] Patients with severe TS will have an elevated RA:RV gradient. In severe PR, elevated RV and RA pressures are noted with a widened pulmonary artery pulse pressure.

Echocardiographic Assessment

Assessment of the tricuspid valve (TV) anatomy on transthoracic echocardiogram (TTE) can be challenging, though the anterior leaflet can be readily evaluated on parasternal long RV inflow and apical 4 chamber views.[3] Assessment of severe TR with continuous wave Doppler demonstrates a dense triangular shape. With severe secondary TR, annular dilation and a large central jet are present. In primary severe TR due to leaflet perforation, an eccentric jet may be noted. Systolic flow reversal in the hepatic veins and a plethoric inferior vena cava (IVC) are also characteristic of severe TR. Tricuspid stenosis can be evaluated in a similar manner to MS and is considered severe if the mean gradient is greater than 5 millimetre Hg.[36–38] Pulmonic stenosis is severe when peak and mean gradients are greater than 64 millimetre Hg and 35 millimetre Hg, respectively.[34]

Medical Stabilization and Mechanical Support Options

Medical management of severe right sided regurgitant lesions involves diuresis for right heart failure, and the approach is similar for stenotic lesions, though with more caution given preload dependence. When cardiogenic shock occurs

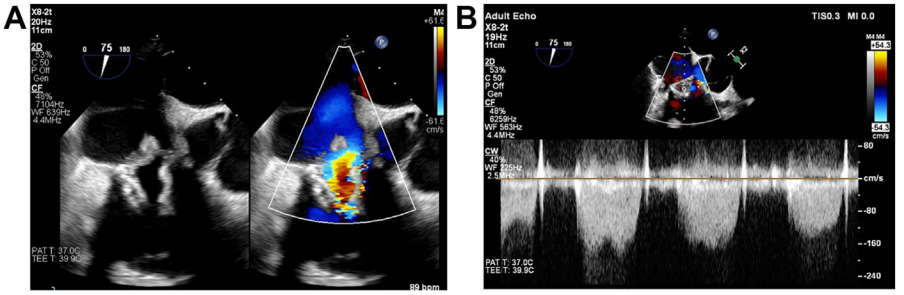

Fig. 12. 37-year-old with a history of tricuspid valve replacement for infective endocarditis presents with fevers and right-sided heart failure. (*A*) Transesophageal echo with a focus on the bioprosthetic tricuspid valves shows associated echodensities in keeping with vegetations. Color Doppler shows aliasing of flow across the tricuspid bioprosthesis and no significant TR. (*B*) Continuous wave Doppler across the bioprosthetic tricuspid valve shows severe TS with peak and mean gradients of 16 and 10 mm Hg, respectively.

from RV failure, inotropes can be used. MCS options are limited, though VA-ECMO will support end-organ perfusion. Percutaneous right ventricular assist devices are also available even though severe stenotic right-sided lesions are a contraindication to their use.

Definitive Repair

For severe TR, TV repair is preferred to replacement when feasible (**Figs. 11** and **12**, Videos 29–31), especially in the setting of endocarditis, where the risk of re-infection is high.[38,39] In general, isolated TV surgery is higher risk compared to surgery for another primary indication with concomitant TV repair. Hemodynamic factors that increase surgical risk include pulmonary hypertension and a decreased cardiac index. When surgical risk is prohibitive, transcatheter approaches can be considered.[40]

Isolated pulmonary stenosis (PS) can be treated with balloon valvuloplasty with reasonable long term outcomes.[41,42] In patients with prior surgery, a transcatheter pulmonic valve can be used within

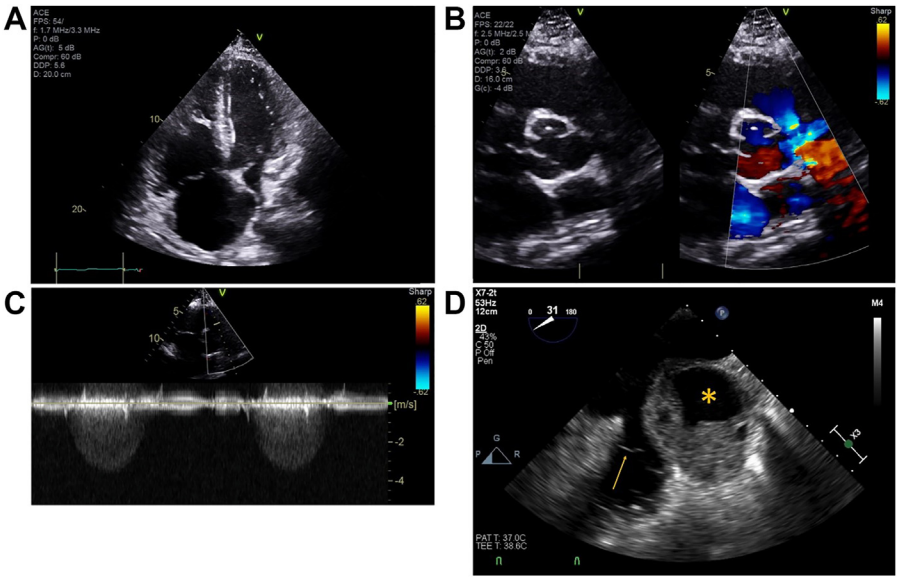

Fig. 13. 57-year-old with a history of coronary artery disease and prior coronary artery bypass grafting presents with cardiogenic shock. (*A*) Apical four chamber shows a dilated and dysfunctional right ventricle. The interatrial septum is also fixed in the left atrium suggesting elevated right atrial pressures. (*B*) Short axis view at the level of the aortic valve shows turbulent flow on color Doppler in the right ventricular outflow tract. (*C*) Continuous wave Doppler across the right ventricular outflow tract demonstrates a late peaking systolic velocity with a peak gradient of 51 mm Hg consistent with significant stenosis. (*D*) On transesophageal echocardiogram, there is a partially thrombosed saphenous vein graft aneurysm (*) extrinsically compressing the proximal pulmonary artery immediately distal to the pulmonic valve (*arrow*).

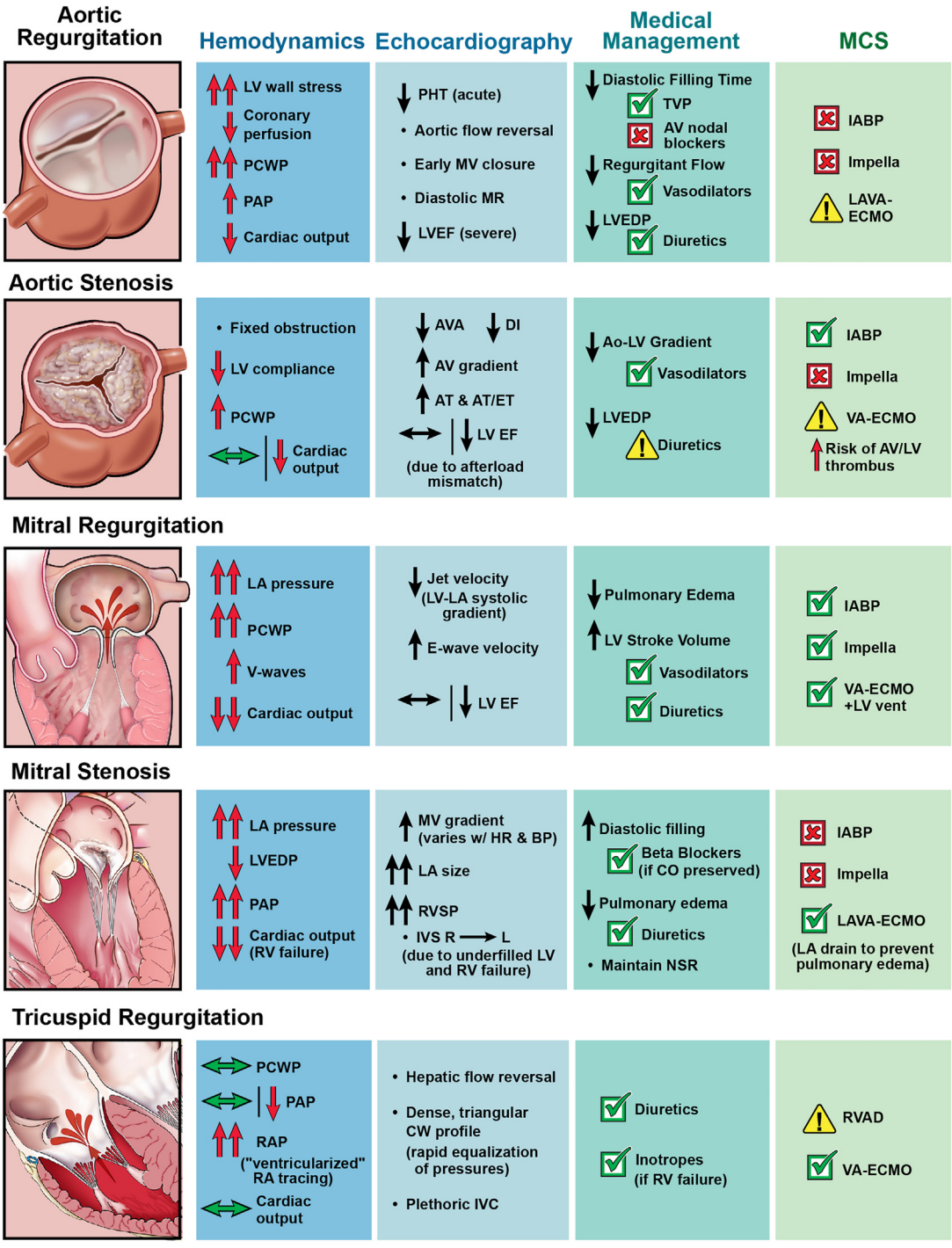

Fig. 14. Central illustration describing the main echo and hemodynamic findings in each valvular lesion, as well as the guiding principles in medical and device management. Green check mark means "clearly beneficial," yellow sign means "use with caution", and red circle indicates "contraindicated". AT, acceleration time; AVA, aortic valve area; CW, continuous wave; ET, ejection time; IABP, intra-aortic balloon pump; IVC, inferior vena cava; LAVA-ECMO, left atrial venoarterial extracorpeal membranous oxygenation; LVEDP, left ventricular end diastolic pressure; LVEF, left ventricular ejection fraction; MR, mitral regurgitation; MV, mitral valve; NSR, normal sinus rhythm; PAP, pulmonary artery pressure; PCWP, pulmonary capillary wedge pressure; PHT, pressure half time; RAP, right atrial pressure; RVAD, right ventricular assist device; RVSP, right ventricular systolic pressure; TVP, temporary venous pacing. (Reprinted with permission, Cleveland Clinic, Cardiology Graphics and Design © 2023. All Rights Reserved.)

a bioprosthetic valve.[43] However, little data are available regarding the optimal management of patients in cardiogenic shock due to PS or extrinsic stenosis of the right ventricular outflow tract (**Fig. 13**, Videos 32–34).

SUMMARY

An understanding of distinctive hemodynamic and echocardiographic features of acute valvular emergencies is essential to guiding medical and device management in the CICU (**Fig. 14**). With acute severe AR, there is a dramatic increase in LVEDP with increased wall stress and decreased coronary perfusion. The result is reduced left ventricular systolic function and pulmonary edema. Echocardiography findings include diastolic aortic flow reversal, a short PHT, early mitral valve closure and diastolic MR, as well as a reduced LVEF. In AS, a fixed obstruction leads to a noncompliant LV. In addition to a low AVA and DI, echocardiography will show a prolonged AT and increased AT/ET ratio. With severe or prolonged afterload mismatch, LVEF may be decreased.

Acute severe MR is devastating due to a precipitous increase in left atrial pressure without prior remodeling of left-sided chambers. Given the decreased LV-LA gradient that can occur with an increased LA pressure and low systolic blood pressure, the MR jet on CW Doppler may be low velocity. In the setting of cardiogenic shock, LVEF may be normal or hyperdynamic. MS results in an increased LA pressure with a normal or low LVEDP. Echocardiography shows increased MV gradients, but especially in the CICU, it is important to remember that MV gradients are dependent on flow (stroke volume) and heart rate. When shock from MS develops, related RV failure is often present with associated accentuated interventricular interaction. Severe TR rarely presents with cardiogenic shock, though this can occur if there is associated pulmonary hypertension or RV failure. On echocardiography, if the orifice is large, severe TR can appear as laminar on color flow mapping and have a low velocity and triangular profile on CW Doppler.

CLINICS CARE POINTS

- Comprehensive assessment of acute valvular emergencies involves a focused physical and echocardiographic examination, with the supplementation of invasive hemodynamics in select cases to guide both diagnosis and management.
- The underlying etiology of the valvular lesion is crucial in determining the appropriate temporizing medical therapy and definitive repair. A transesophageal can often be useful in better elucidating the underlying mechanism.
- Medical management should be tailored to mitigate the hemodynamic insult from the valvular lesion with the end goal of stabilizing the patient for a definitive repair.
- Decision making regarding MCS should be approached with careful consideration of the underlying hemodynamics and valvular anatomy.
- Acute valvular emergencies cannot be remedied by medical management or MCS alone and ultimately require a definitive repair, either transcatheter or surgical.

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DISCLOSURE

The authors have nothing to disclosures.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found online at https://doi.org/10.1016/j.ccl.2024.02.009.

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